A DISSERTATION ON

ETIOLOGY, CLINICAL PROFILE AND OUTCOME OF COMA IN CHILDREN

M.D (BRANCH VII)

PAEDIATRIC MEDICINE

MARCH 2009

THE TAMILNADU
DR.MGR.MEDICAL UNIVERSITY
CHENNAI, TAMILNADU

CERTIFICATE

This is to certify that the dissertation entitled **"Etiology, Clinical profile and outcome of Coma in children"** submitted by **Dr.S. Pushparani** to the Faculty of Paediatrics, The Tamil Nadu Dr.M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.D. Degree Branch VII (Pediatrics) is a bonafied research work carried out by her under our direct supervision and guidance.

Dr.P.Amutha Rajeshwari,M.D.,DCH.,

Professor and Head of the Department,
Institute of Child Health & Research Centre,
Madurai Medical College,
Madurai.

DECLARATION

I **Dr. S. Pushparani** solemnly declare that the dissertation titled "**Etiology, Clinical profile and outcome of Coma in children"** has been prepared by me.

This is submitted to the **Tamilnadu Dr.M.G.R.Medical University**, Chennai in partial fulfillment of the rules and regulations for the M.D.Degree Examination in Paediatrics.

Place: Madurai

Date: **Dr. S. PUSHPARANI**

ACKNOWLEDGEMENT

It is with immense pleasure and privilege that I express my heartful gratitude, admiration and sincere thanks to my chief **Prof.Dr.P.AmuthaRajeswari,** Professor and Head of the Department of Pediatrics, for her guidance and support during this study.

I am greatly indebted to my guide and teacher, Rtd **Prof.Dr.D.Meikandan**, Neurologist and Professor of Pediatrics, for his supervision, guidance and encouragement while undertaking this study.

I express my sincere thanks and gratitude to Prof. **Dr.M.L.Vasantha kumari** for her support and for her guidance, supervision, constant encouragement and support throughout this study.

I wish to express my sincere thanks to my Asst Profs **Dr. S. Venkateshwaran, Dr. S. Nataraja Rathinam** for their valuable suggestions and support.

I would like to thank **Prof.Dr.S.Rajasekaran, Prof.Dr.G.Mathevan, Prof. Dr. Chitra Ayyappan and**

Prof. Dr, R . A. Sankarasubramaniam who guided me to a great extent. I also thank all the members of the Dissertation Committee for their valuable suggestions.

I gratefully acknowledge the help and guidance received from **Asst. Prof. Dr. K. Mathiarasan** at every stage of this study.

I also express my gratitude to all my fellow postgraduates for their kind cooperation in carrying out this study and for their critical analysis.

I thank the Dean and the members of Ethical Committee, Government Rajaji Hospital and Madurai Medical College, Madurai for permitting me to perform this study.

I thank all the parents and children who have ungrudgingly lent themselves to undergo this study without whom this study would not have seen the light of the day.

CONTENTS

INTRODUCTION

KOMA in GREEK means DEEP SLEEP.

Coma is a medical emergency and may constitute a diagnostic and therapeutic challenge for the intensivist.

Coma indicates prolonged state of unarousable sleep and disturbance of consciousness usually resulting from lesions involving the reticular formation of brain stem, the hypothalamus and connection with the cerebral hemispheres.

Acute nontraumatic coma accounts for about 10- 15% of the hospital admissions and is associated with significant mortality.

Coma can result from a wide range of primary etiologies, of which the major causes are infection, hypoxia, epilepsy, accidental and non accidental injury .

Etiology of coma and clinical status at the time of presentation are the important predictors of outcome.

Impairment of the conscious level is objectively graded according to Glasgow coma scale(GCS) and is used to monitor the progress. Although it has serious limitations in infants and young

children, a Modified form of the GCS (MGCS) has been used in them to assess the severity, even though only a very few studies are available to support its use in pediatric coma as a whole.

Although necessarily limited, a careful neurological examination is very important in an unconscious children. Posture, pupil size and reactivity, spontaneous eye ball movements and the reflex eye movements helps to determine the level of structural damage and depth of coma.

ANATOMIC SUBSTRATE OF ALERTNESS:

Consciousness is the complex interplay between cerebral cortex and sub cortical structures i.e., Diencephalon, Midbrain, upper Pons. In general the maintenance of consciousness depends on the interaction between the Ascending Reticular Activating system and the cerebral hemispheres. The ARAS lies in the paramedian tegmental region of the posterior portion of the Pons and the Midbrain. It extends from the superior half of the Pons through the Midbrain to the posterior portion of the Hypothalamus to the thalamic reticular formation.

The Median Longitudinal fasciculi, which connects the abducens and the oculomotor nuclei, and the oculomotor and trochlear nuclei themselves are situated amid the neurons of the pontine and midbrain portions of the ARAS. Thus when unresponsiveness is caused by brainstem damage, the lesion affects the mechanisms of ocular motility as well, and its location can be determined by abnormal patterns of ocular motility.

A better understanding of causes and outcome is essential to help improve the approach and to plan rational management of nontraumatic coma.

This study was conducted to assess the various etiologies, and to assess the relation between MGCS, the various clinical factors that helps to predict the outcome of coma.

APPROACH

HISTORY

↓

DETAILED GENERAL & SYSTEMIC EXAMINATION

↓

COMPLETE NEUROLOGICAL EXAMINATION

↙ ↘

Initial laboratory studies
CBC, ESR, CRP
Blood glucose
Urea & creatinine
Sr. Electrolytes
Sr. Calcium
Blood cultures
CSF analysis
Liver function tests
Mantoux

↓

Subsequent laboratory tests
EEG
Toxicology screening - urine, blood
ABG
Blood coagulation profile
Metabolic screening
Sr. ammonia, lactate levels
Blood anticonvulsant levels

Radiographic studies
Ultrasound
Head CT
Head MRI/ MRA

Literature on pediatric non-traumatic coma is rather inconclusive, as there are few systematic studies, and most of these are retrospective. Very little information is available particularly so from developing countries including India.

INCIDENCE:

The incidence of coma in children under 16 years varies from place to place. Wong CP et el study[2] on "Incidence etiology and outcome of coma", the incidence of coma is 30.8 per 100 000 children under 16 per year (6.0 per 100 000 general population per year). The age specific incidence was notably higher in the first year of life (160 per 100 000 children per year).

In Sofiah A., Hussain I. H. M. et el [3] studies on nontraumatic coma, 69% were due to infection, 13% were due to toxic metabolic causes, 5% were due to hypoxic ischaemic insults, 3.5% had intracranial haemorrhage, 7.8% were due to miscellaneous causes and in 1.7% the cause was unknown. Age of onset and sex did not significantly affect outcome.

Stevens and Bharadwaj et el [4] reviewed all the currently available data on the etiology, diagnosis, and outcome of coma and proposed an evidence-based approach for the clinical management of the comatose patient. They also wrote the neurologic prognosis is determined by the underlying etiology and may be predicted by the combination of clinical signs and electrophysiological tests.

Seshia SS, Johnston B, Kasian G et el [5] showed analysis of clinical variables recorded early in the comatose state can provide predictive information and stepwise discriminant analysis may be one method of determining the most likely outcome for individual cases.

According to a study conducted by Lohr Junior A et el [6],done to study the etiology, morbidity and mortality of coma in children, 31 (29.8%) of the cases were due to meningo-encephalitis,24 (23.1%) to an epileptic condition,

19 (18.3%) weretoxic-metabolic, 16 (15.4%) to intra-cranial hypertension, 7 (6.7%) to shock/anoxia, 4 (3.8%) to an indeterminate etiology and 3(2.9%) were miscellaneous.

Arun Bansal et el[7] analysed 100 consecutive cases of non traumatic coma. He found etiology of coma in 60% cases was CNS infection (tubercular meningitis - 19, encephalitis - 18, bacterial meningitis -16,others-7); other causes were toxic-metabolic conditions (19%), status epilepticus(10%), intracranial bleed (7%), and miscellaneous (4%).

According to a study conducted by Aswati. et el[8] from Department of Paediatrics, King George's Medical College, Lucknow, Uttar Pradesh, to study the value of MGCS to predict the mortality in patients with acute CNS infection along with other clinical variables, they found that MGCS can be used to predict discharge in patients with acute infective disorders of the central nervous system within 24 hours of hospitalization. The scale is simple, can be applied at the bedside and does not depend on any investigations.

A Tatman & A Williams et el [9] showed in their study that James' adaptation of the Glasgow coma scale (JGCS) was designed for young children. Intubated patients are not allocated a verbal score, however, so important changes in a patient's conscious level may be missed. A grimace score [33] was therefore developed and assessed for use in intubated children.

Nayana P.C. Prabha et el [10] in their study noted that low total MGCS was found to be associated with adverse outcome. They also observed a relationship between brainstem reflexes and poor outcome. Absence of one or more reflexes predicted adverse short term outcome. They also noted that etiology did not affect the short term outcome. They concluded ocular, motor response scores and brainstem reflexes are more predictive of short term outcome than MGCS.

Bolton et el [14] proposed that clinical examination combined with the results of Electro Encephalography (EEG) and somatosensory evoked potentials can be used to establish an early, definitive prognosis in a significant proportion of patients in anoxic coma.

DEFINITIONS [33,34,35]

CONSCIOUSNESS:

Consciousness defined as a state of awareness of self & surrounding.

ALERT:

Alertness is the normal state of arousal.

DELIRIUM:

Delirium is agitated confusional stage, in which the child responds incoherently and many times violently. The child will respond continuously without slipping back to sleep.

DROWSY:

The child is sleeping but arousable by ordinary stimulus or a loud call. Once awake, the child responds appropriately without further stimulation, but goes into sleep again if left alone.

STUPOR:

Stupor is the state of unresponsiveness from which the subject can be aroused only by vigorous (painful) and repeated stimuli. Soon after the removal of stimulus, the child slips into sleep.

OBTUNDATION:

It is a stage between drowsiness and stupor. Here the child may be awakened by a strong stimulus other than pain (loud call with simultaneous shaking). The child will respond inappropriately to the stimulus.

COMA :[37]

"The patient who appears to be in asleep and at the same time is incapable of being aroused by any external stimulus or by his inner needs" is said to be in coma.

IMPORTANT CAUSES OF ACUTE ENCEPHALOPATHY [33]

INFECTIOUS & PARA INFECTIOUS ENCEPHALOPATHIES:

Meningitis

Cerebral abscess

Primary viral encephalitis

Post infectious encephalitis

Cerebral malaria

Acute Disseminated EncephaloMyelopathy (ADEM)

HYPOXIC ISCHEMIC ENCEPHALOPATHIES

Status epilepticus

Near drowning

Near miss Sudden Infant Death syndrome

Post cardiac arrest, Cardiac arrhythmias, Hypotension

Disseminated Intravascular Coagulopathy

Hypoglycemia

Vitamin & other cofactor deficiencies

EXOGENEOUS TOXINS

Neem oil/Camphor

DRUGS

Antihistamines, Antidepressants, Hypnotics and sedatives

Analgesics

Antiepileptics

Anti-inflammatory drugs

Antimetabolites

ILLICIT SUBSTANCES

Alcohol , Cannabis, Cocaine,Opiates

ENVIRONMENTAL TOXINS

Carbon monoxide, Phosphates, DDT, Iron, Lead, Pesticides, venom.

HYPOTHERMIA

HEAT STROKE

HYPERTENSION

ENDOGENEOUS AGENTS

Water intoxication

Acidosis and Alkalosis

ENDOCRINE DISORDERS

Diabetic Keto Acidosis, Hypoglycemia, Hypothyroidism, Hyperthyroidism, Hypoadrenalism, Hypopituitarism, Hypoparathyroidism, Hyperparathyroidism

ORGAN FAILURE

Hepatic failure

Renal failure

Reye syndrome

INBORN ERRORS OF METABOLISM

Urea cycle defects, Fatty acid oxidation defects,

Mitochondrial disorders

CEREBRO VASCULAR DISEASE

NONCONVULSIVE STATUS EPILEPTICUS

GRADES OF COMA

Stage 1 or stupor:

The patient can be aroused briefly and shows verbal or motor response to stimuli.

Stage 2 or light coma:

The patient cannot be aroused easily, except with painful stimulus.

Stage 3 or deep coma:

There is no response to painful stimuli. The limbs may be

kept in primitive reflex posture. The limbs may be in decorticate or decerebrate posturing.

Stage 4 or brain death:

All cerebral functions are lost. Pupillary reflexes are lost. There is no spontaneous respiratory effort, but local spinal reflexes are preserved.

BEHAVIORAL STATES CONFUSED WITH COMA

1. LOCKED IN SYNDROME:

Locked in syndrome refers to a condition in which the patient is mute and motionless (deefferented) but remains awake, alert, aware of self and capable of perceiving the sensory stimuli due to bilateral ventral pontine lesions. Here the subject is only able to move eyes vertically or blink or both, though the horizontal eye movements are impaired (PPRF).

This syndrome is due to Pontine infarction following basilar artery thrombosis, Pontine hemorrhage or tumor or central Pontine myelinolysis. The Reticular Activating system remains

intact.

2. PERSISTENT VEGETATIVE STATE

Persistent vegetative state is defined as vegetative state persisting for atleast one month after acute traumatic or non traumatic brain injury or in patients with degenerative or metabolic disorders or developmental malformations.

It is characterized by lack of awareness of self and external stimuli, accompanied by sleep-wake cycles, with preservation of vital vegetative functions such as cardiac function, respiration, and maintenance of BP. Spontaneous movements may occur and eyes may open in response to external stimuli but subject does not speak or obey commands.

3.ABULIA

It is a state of severe apathy in which subject has blunting of feeling, drive, mentation and behavior so they neither speak nor move spontaneously.

4. CATATONIA

It is the state of muteness with dramatically decreased motor activity. The maintenance of body posture with preserved ability to sit or stand distinguishes from organic stupor.

5. PSEUDOCOMA(PSYCHOGENIC UNRESPONSIVENESS)

In this state the subject appear comatose but have no structural, metabolic, toxic causes.

ENCEPHALOPATHY:

Encephalopathy is a disorder of consciousness and applied to comatose state or continuum of worsening of altered states of consciousness from fully alert to deep coma.

Arousal is impaired in encephalopathy and absent in coma.

COMA SCALES

1. CLINICAL STAGING SYSTEM
2. GLASGOW COMA SCALE
3. CHILDREN COMA SCALE
4. MODIFIED CHILDREN COMA SCALE

GLASGOW COMA SCALE

Total score is 15 and the minimum score is 3. Score less than 8 require aggressive management. GCS provides a rapid assessment of cerebral cortical function.

GLASGOW COMA SCALE AND PEDIATRIC COMA SCALE [9]

Sign	GCS	PCS	Score
Eye opening	Spontaneous To command To pain None	Spontaneous To sound To pain None	4 3 2 1
Verbal response	Oriented	Age-appropriate vocalization, smile, or orientation to sound	5
	Confused, disoriented	Irritable, consolable, uncooperative, aware of the environment	4
	Inappropriate words	Irritable, inconsistently consolable	3
	Incomprehensible sounds	Inconsolable, unaware of the environment, restless, agitated	2
	None	None	1

Motor response	Obeys commands	Obeys commands, spontaneous movements	6
	Localizes pain	Localizes pain	5
	Withdraws	Withdraws	4
	Abnormal flexion to pain	Abnormal flexion to pain	3
	Abnormal extension to pain	Abnormal extension to pain	2
	None	None	1
	Best total score		15

- Data from: Teasdale, G and Jennett, B. Assessment of coma and impaired consciousness. A practical scale. Lancet 1974; 2:81.
- Data from: Simpson, D and Reilly, P. Pediatric coma scale (letter). Lancet 1982; 2:450.(9

Glasgow coma scale which is widely applicable to adults depends on higher integrative function which is not present in infants or very young children.

Modification of Glasgow coma scale alters the best verbal response to following:

MODIFIED COMA SCALE FOR INFANTS & CHILDREN LESS THAN 4 YEARS [33]

Coos and babbles - 5
Irritable cries - 4
Cries to pain - 3
Moans to pain - 2
None - 1

The best possible response changes according to language

development so that the maximum total score is adjusted to reflect maturation as follows:

Birth to 6 months	09
6months to 12months	11
1year to 2year	12
2year to 5year	13
More than 5 year	14

CLINICAL STAGING IN ENCEPHALOPATHY [35]

STAGE 1

Lethargic
Follows commands
Pupils reactive
Breathing normal
Normal muscle tone

STAGE 2

Combative
Inconsistently following commands
Pupils sluggish
May hyperventilate
Reflexes inconsistent

STAGE 3

Comatose
Occasional responds to command
Eyes may deviate
Irregular breathing
Decorticate posturing

STAGE 4

Comatose
Respond only to pain
Weak pupillary response
Very irregular breathing
Decerebrate posturing

STAGE 5

Comatose
No response to pain
No pupillary response
Require mechanical ventilation
Flaccid state

A simple bedside assessment can be made using the AVPU

SCALE:

ALERTNESS

RESPONSE TO **V**ERBAL COMMAND

RESPONSE TO **P**AIN

UNCONSCIOUS

SIGNS WITH LOCALISING VALUES IN COMA [34, 36, 26]

For localization of structural lesions and to assess the prognosis the following examinations are helpful

1) State of consciousness

2) Pattern of breathing

3) Pupillary size & reactivity

4) Eye movements

5) Motor movements & involuntary movements

MANAGEMENT:

Children in coma need repeated reassessment with particular attention to pupil size, reactions, reflex eye movements, motor response, fundus and skilled neurological care.

Cerebral perfusion pressure and cerebral blood flow should be maintained and monitoring of ICP can be useful in head trauma, intracranial infection and certain metabolic encephalopathies.

Control of seizures and specific treatment as per etiology like antibiotics, antidotes, metabolic corrections should be made appropriately.

INVESTIGATIONS :

1. Complete blood count
2. Blood – Glucose, S. Calcium
3. Markers of inflammation (ESR, CRP)

4. Blood & urine osmolarity
5. Coagulation profile
6. Bacteriological & virological studies (Culture, serology, PCR)
7. Mantoux
8. Imaging studies – USG, CT, MRI
9. Arterial Blood Gas analysis
10. Plasma Ammonia, Plasma lactate – To R/o inborn errors of metabolism
11. Urine & Blood Toxicology – for drugs like anti-histamines, anticholinergics, antidepressants, hypnotics & sedatives, analgesics, anti epileptics, antimetobolites, alcohol, cannabis, cocaine, opiates.
12. Blood – Anticonvulsants level
13. Urine metobolic screening - To R/o Aminoacidopathies, organic acidurias, Urea cycle defects, mitochondrial disorder
14. CSF Analysis
15. Liver function test
16. TFT & other endocrine investigations – to R/o DM, Hypoglycemia, DKA, Hypo & hyperthyroidism, Hypo & Hyperparathyroidism,

hypopituitarism, hypoadrenalism.

TREATMENT :

- ABC - maintain airway, breathing and circulation
- Cervical immobilization : It should be employed where there is any possibility of cervical spine trauma.
- Intubate the patient if GCS is 8 or less than 8
- Avoid neck flexion, which may obstruct jugular venous return.
- Elevate head end of the bed to 15-30^{O} with the head in neutral position to reduce venous outflow pressure.
- Sedation and neuromuscular blockade to avoid valsalva maneuvers from coughing which is common with an endothracheal tube in place.
- Maintain normal or high normal blood pressure with isotonic fluids and inotropic support, if needed.
- Maintain body temperature normal to slightly hypothermic to reduce the whole body and cerebral metabolism.
- Inj. Mannitol 0.25 – 0.5 gm/kg - every 6th hourly
- Steroids – useful in reducing focal edema around mass lesions and in

pyogenic meningitis.

- Ventricular Catheterization – allows most accurate measurement of ICP and therapeutic drainage of CSF to lower ICP.

MODERATE HYPERVENTILATION - Leads to cerebral vasoconstriction and a reduction in cerebral blood flow and volume, decreasing ICP.

  - Maintain PCO2 at 25-35 mm Hg
  - More aggressive or prolonged hyperventilation may produce cerebral ischemia and should be avoided.

- Bladder care : Catheterize the bladder
- Bowel Care : Suppositories and oral lactulose
- Eye Care : Artificial tear drops and topical antibiotics
- Back Care : Change the position of the patient frequently and use skin hardening solutions like spirit.
- Administer specific treatment according to the etiology

LONG TERM MANAGEMENT AND PROGNOSIS :

- Duration of coma is the important indicator of long term disability

- In EEG - Indicators for poor outcome are
    i. Low-voltage undifferentiated tracings
    ii. Burst suppression
    iii. Electro cerebral inactivity
- Once mechanical ventilation can be discontinued, circulatory function has stabilized and ICP has normalized, early rehabilitation interventions like various sensory stimulation techniques, physiotherapy, occupational therapy and speech therapies should be initiated.

AIM OF THE STUDY

1) To know the different etiologies of coma in children between 1 month to 12 years.

2) To determine the clinical signs predictive of outcome

MATERIALS AND METHODS

STUDY CENTRE:

The study was conducted in the Institute of Child Health and Research Center, Government Rajaji Hospital, Madurai.

STUDY PERIOD:

The study was carried out prospectively from December 2006 to May 2008.

STUDY DESIGN:

Prospective observational study

STUDY POPULATION:

Children admitted in Govt. Rajaji Hospital, Madurai medical college, Madurai.

SAMPLE SIZE : 180 Children

INCLUSION CRITERIA

Children between 1 month- 12 yrs fitting with the definition of coma

EXCLUSION CRITERIA

1) Age < 1 month & >12 yrs
2) Traumatic coma
3) Child with neuro developmental delay, any other pre-existing neurological illness.

CONFLICT OF INTEREST : Nil

FINANCIAL SUPPORT : Nil

ETHICAL COMMITTEE CLEARANCE: Obtained

METHODOLOGY:

For all children admitted with the above mentioned criteria, a detailed history, general examination and a focused neurological examination (brainstem reflexes including pupillary reactivity) were made. MGCS were assessed every 12th hourly from the time of admission upto 48 hrs. In case of children who were intubated or who developed respiratory failure secondary to neurological condition, the best verbal response is taken as grimace. Etiology of coma was determined on basis of clinical history, examination and relevant laboratory investigations. All the children were followed up till discharge or death. The outcome was recorded as survived or died, and among those who survived, as normal, or with sequelae.

STATISTICAL METHODS:

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using Epidemiological Information Package (EPI 2002).

Using this software, frequencies, percentages, means, standard deviations, Chi- square, 'p' and coefficient of correlation values were calculated. Kruskul Wallis Chi- square test was used to test the significance of difference between quantitative variables and Yate's test for qualitative variables. A 'p' value less than 0.05 is taken to denote significant relationship. If the coefficient of correlation (r) is more than 0.5 then the two variables are taken to be correlated.

ANALYSIS:

Data will be spread in excel spread sheet and analysed using simple descriptive statistics.

CASE DEFINITIONS

PYOGENIC MENINGITIS [24]:

Acute febrile encephalopathy with culture positive Cerebro Spinal Fluid or presence of the 2 of the following abnormalities in CSF – polymorphonuclear leukocytes/ glucose <40mg/dl or 50% of blood sugar/ micro organisms seen by gram staining.

TUBERCULOUS MENINGITIS:

Child with features of meningitis, with CSF pleocytosis with lymphocyte predominance and organisms seen by AFB staining/ absence of bacteria on direct microscopy, elevated proteins, CT features s/o TB- basal meningitis, obstructive hydrocephalus, prominent cisterns.

VIRAL MENINGOENCEPHALITIS:

Acute febrile encephalopathy with CSF pleocytosis with lymphocyte predominance and absence of bacteria on direct microscopy.

HYPERTENSIVE ENCEPHALOPATHY:

Encephalopathy in association with BP >95th percentile for age and sex with or without retinal changes.

HYPOXIC ISCHEMIC ENCEPHALOPATHY:

Encephalopathy following hypoxic cerebral injury such as near drowning, accidental/ homicidal hanging.

TOXIC ENCEPHALOPATHY:

Encephalopathy following ingestion of toxin containing substances (neem oil, organophosphorus, drugs, kerosene)

HEPATIC ENCEPHALOPATHY [30]:

Spectrum of potentially reversible neuropsychiatric abnormalities seen in patients with liver dysfunction after exclusion of unrelated neurologic and metabolic abnormalities

INTRACRANIAL BLEED:

Children with coma with evidence of bleed on radio imaging of head.

RESULTS

The study population consisted of 180 children with acute non-traumatic coma whose average age was 3 years and 5 months.

TABLE - 1

AGE GROUP

Age Group	Cases	
	No.	%
< 1 year	51	28.3
1-5 years	86	47.8
6 – 12 years	43	23.9
Total	180	100
Mean	3.46 yrs	
S.D.	3.16 yrs	

51 were less than 1 yr of age, 86 were between 1 to 5 yrs, 43 were between 6 to 12 yrs with a mean age of 3.46 yrs.

SEX DISTRIBUTION

Of the 180 children, 106 were males and 74 were females.

TABLE - 2

SEX	Cases	
	No.	%
Males	106	58.9
Females	74	41.1
Total	180	100

ETIOLOGY OF COMA : TABLE - 3

S. No	Diagnosis	No of cases	% of cases among the total
I	Intracranial infection A) Pyogenic Meningitis B) TBM C) Viral Meningo encephalitis D) Dengue E) Cerebral Malaria F) ADEM	99 52 18 20 2 2 5	55.2%
II	Vascular a) Intra cranial Haemorrhage i) Parenchymal ii) Subdural iii) Sub thalamic b) Thrombosis c) CVT d) Infarction – vasculitis	17 13 11 1 1 2 1 1	9.4%
III	Encephalopathy A) Toxic encephalopathy (Neem oil poisoning, Drugs, OPC, Cypermethrin) B) Metabolic i) Hepatic encephalopathy Wilson disease Leptospirosis Chronic liver disease ii) Diabetic coma C) Hypoxic encephalopathy i) Status epilepticus ii) Cobra bite iii)Near drowning D) Hypertensive encephalopathy i) CRF ii) AGN	59 16 - - 19 15 3 2 10 4 21 18 1 2 3 2 1	32.7%
IV	Idiopathic	5	2.7%
	Total	180	100%

CLINICAL PROFILE

MODIFIED GLASGOW COMA SCALE AT 24 & 48 HOURS

TABLE - 4

MGCS Score	MGCS Score at			
	24 hours		48 hours	
	No.	%	No.	%
3	62	34.4	21	16.8
4-6	77	42.8	38	30.4
7-8	21	11.7	17	13.6
9-12	15	8.3	30	24.0
13-15	5	2.8	19	15.2
Total	180	100	125	100
Mean SD	5.37 2.77		7.61 3.83	

The MGCS at 24 hours was < 8 in 160 patients and between 9- 12 in 15 and 13 – 15 in 5 patients with a mean score of 5.37. The MGCS at 48 hours was <8 in 76, between 9- 12 in 30 and >12 in 19 with a mean score of 7.61.

PUPILLARY REFLEX

TABLE - 5

Pupillary Reflex	Cases	
	No.	%
Reactive	136	75.6
Non Reactive	44	24.4
Total	180	100

Of the 180 studied, pupillary reflexes were preserved in 136 patients and absent in 44 cases.

BRAINSTEM REFLEXES

TABLE - 6

Brain Stem Reflex	Cases	
	No.	%
Present	110	61.1
Absent	70	38.9
Total	180	100

Brainstem reflexes were preserved in 110 cases out of 180 cases in our study.

WITH INTRACRANIAL HYPERTENSION

ICT was assessed clinically by the presence of Cushing reflex, papilloedema on fundus examination.

TABLE - 7

Raised ICT	Cases	
	No.	%
Normal	148	82.2
Abnormal	32	17.8
Total	180	100

Of the 180 studied 32 had features of raised ICT.

REQUIRING VENTILATORY SUPPORT

TABLE - 8

Ventilator	Cases	
	No.	%
Yes	25	13.9
No	155	86.1
Total	180	100

Of the 180 studied, ventilatory support was needed in 25(13.9 %) cases.

DURATION OF COMA

TABLE - 9

Duration of Coma	Cases	
	No.	%
< 24 hours	89	49.7
25 – 48 hours	37	20.7
49 – 72 hours	36	20.1
> 72 hours	18	9.5
Total	180	100
Mean SD	43 hours 37.4 hours	

The duration of coma was <24 hours in 89, between 25 – 48 hours in 37, between 49- 72 hours in 36. In 18 cases coma persisted for >72 hours. The mean duration of coma was about 43 hours.

OUTCOME

TABLE - 10

Outcome	Cases	
	No.	%
Alive	78	43.3
Death	102	56.7
Total	180	100

Among the 180 cases studied 78 (43.3%) survived, and 102 (56.7%) cases died. Of the 102 cases , 36 died within 24 hours of admission, 55 died between 24- 48 hours and 11 cases died after 48 hours.

MORTALITY PATTERN
TABLE - 11

S.No	Diagnosis	No.of Deaths	% of deaths among total deaths
I	**Intracranial infection(99)**	59	58.6 %
	A) Pyogenic Meningitis (52)	39	
	B) TBM (18)	6	
	C) Viral Meningoencephalitis(20)	10	
	D) Dengue (2)	1	
	E) Cerebral Malaria (2)	1	
	F) ADEM (5)	2	
II	**Vascular(17)**	10	9.8 %
	a) Intra cranial Haemorrhage (13)	9	
	i) Parenchymal(11)	7	
	ii) Subdural(1)	1	
	iii) Sub thalamic(1)	1	
	b) CVT(1)	1	
III	**Encephalopathy(59)**	31	30.3 %
	A) **Toxic** encephalopathy (16)	6	
	Neem oil poisoning (8)	4	
	Drugs – CBZ (3)	1	
	OPC (1)	1	
	B) **Metabolic**(19)	11	
	i) Hepatic encephalopathy (15)	10	
	Wilson disease (3)	3	
	Chronic liver disease (10)	7	
	ii) Diabetic coma (4)	1	
	C**) Hypoxic** encephalopathy (21)	13	
	i) Status epilepticus (18)	12	
	ii) Cobra bite (1)	1	
	D) **Hypertensive** encephalopathy(3)	1	
IV	Idiopathic (5)	2	1.9 %
	Total	102	100

The mortality was high among intracranial infection (58.6%).

RELATIONSHIP BETWEEN VARIOUS PARAMETERS AND OUTCOME:

AGE & OUTCOME

TABLE - 12

Age	Outcome	
	Alive	Dead
Mean	2.75	4.00
S.D.	2.62	3.43
'p'	0227 Significant	

There is a statistically significant co-relation between the age and outcome with mean age for death about 4 years.

GENDER AND OUTCOME

TABLE - 13

Sex	Outcome			
	Alive		Dead	
	No	%	No	%
Males(106)	52	49.1	54	50.9
Females(74)	26	35.1	48	64.9
'p'	0.0889 Not significant			
Relative risk	1.4			

There is no statistically significant relation between gender and mortality

ETIOLOGY AND OUTCOME
TABLE - 14

Etiology / Diagnosis	Outcome			
	Alive		Dead	
	No	%	No	%
I)Intra cranial Infection (99)	40	41.4	59	58.6
II) Vascular (13)	7	41.1	10	58.9
III)Encephalopathies (63)	28	47.5	31	52.5
IV) Idiopathic (5)	3	60	2	40
"p" Value between				
I & II	0.2701-Not significant			
I&III	0.9712 -Not significant			
I & IV	0.1272 -Not significant			

There is no significant co-relation between the etiology and outcome. The mortality was higher in coma due to vascular pathology, 10 out of 13 cases died (58.9%). The mortality was also higher in intracranial infection 59 out of 99 cases (58.6%)

RELATION BETWEEN MGCS AND OUTCOME AT 24 HOURS

TABLE - 15

MGCS at 24hrs	Alive		Dead	
	No	%	No	%
3 (62)	7	11.3	55	88.7
4 – 5 (77)	37	48.1	40	51.9
6 - 8 (21)	18	85.7	3	14.1
9 – 12 (15)	11	73.3	4	26.7
13 – 15 (5)	78	100	-	-
Total (180)		100	102	100
'p'	0.0001 Significant			

RELATION BETWEEN MGCS AND OUTCOME AT 48 HOURS

TABLE - 16

MGCS at 48 hrs	Alive		Death	
	No	%	No	%
3 (21)	2	9.5	19	90.5
4 – 5 (38)	15	39.5	23	60.5
6 – 8 (17)	13	76.5	4	23.5
9 – 12 (30)	27	90.0	3	10
13 – 15 (18)	16	84.2	3	15.8
Total (125	73	100	52	100
'p'	0.0001 significant			

Death is more common in cases with lower MGCS scores at 24 hr and 48 hrs.

PUPILLARY REFLEX AND OUTCOME

TABLE - 17

Pupillary Reflex	Outcome			
	Alive		Dead	
	No	%	No	%
Reactive (136)	73	53.7	63	46.3
Non Reactive (44)	5	11.4	39	88.6
p value	0.0001 (Significant)			
Relative Risk	4.72			

Non reactive pupils were associated with a higher mortality of 88.6%, (39 out of 44 cases). There is a statistically significant co- relation between pupillary reflex and outcome with a relative risk of 4.72 times.

BRAINSTEM REFLEXES AND OUTCOME

TABLE - 18

Brain Stem Reflex	Outcome			
	Alive		Dead	
	No.	%	No.	%
Present (110)	67	60.9	43	39.1
Absent (70)	11	15.7	59	84.3
"p" Relative Risk	0.0001 (Significant) 3.88			

Absent brainstem reflexes are associated with a high percentage of mortality (84.3%), 59 out of 70 cases. There is statistically significant correlation between absent brainstem reflexes and outcome with a relative risk of 3.88 times.

DURATION OF COMA AND OUTCOME

TABLE - 19

Duration of Coma in hours	Outcome	
	Alive	Dead
Mean	39.39	45.73
SD	34.23	39.6
"p"	0.1785 Non Significant	

The mean duration of coma for alive are 39.39 hours and for the death it is about 34.23 hours. There is no significant correlation between the mean duration of coma and outcome.

RAISED ICT AND OUTCOME

TABLE - 20

With raised ICT	Outcome			
	Alive		Dead	
	No.	%	No.	%
Normal (148)	68	45.9	80	54.1
Abnormal (32)	10	31.3	22	68.8
"p"	0.1853 (Not Significant)			
Relative Risk	1.42			

There is no statistically significant correlation between raised ICT and outcome.

DISCUSSION

It is a well known fact that the prognosis in coma depends on its severity. Assessing the severity of coma by subjective, poorly defined terms such as stupor, semi-coma, deep coma was ineffective in predicting the outcome and there was a great deal of inconsistency when different observers carried out the assessment [17].Glasgow Coma Scale (GCS) Score reflects the integrity of cerebral functions. Eye movements (in response to vestibularstimulation), pupillary responses, and corneal responses primarily express functions regulated by the brain stem.

The Glascow coma scale is a standardized system developed initially in traumatic coma to assess the degree of coma and to identify the seriousness of brain injury in relation to the outcome [9].It has gained widespread use as it is highly reproducible, can be quickly performed at the bedside and provides useful information on the progress and prognosis of comatose individual [18, 19].

One limitation of the Glasgow coma scale is that only a few studies are available regarding its usefulness in non-traumatic pediatric coma as a whole [15].

Another drawback of GCS score is loss of information, which occurs due to the summation of individual scores [20].

In comatose patients, evidence of widespread damage of brain stem or cerebral hemispheres at onset usually predicts death or severe disability. Therefore clinical signs that reflect extent and severity of dysfunction of cerebral hemispheres and/or brain stem were studied.

This study included 180 children in the age group of 1 month to 12 yrs.

51 were less than 1 yr of age, 86 were between 1 to 5 yrs, 43 were between 6 to 12 yrs with a mean age of 3.46 yrs. Of the 180 children, 106 (58.9%) were males and 74 (41.1%)were females.

Incidence of coma was higher in males when compared to females.But this is incontrast to a study by Seshia[5] and Seshia et el who did not observe any significant difference in the incidence of coma between the two sexes.

ETIOLOGICAL PROFILE:

With regard to etiology, Intracranial infection accounts for about 99 (55.2%) cases which included 52 cases of pyogenic meningitis (52.5%), 18 cases of tuberculous meningoencephalitis (18%), 22 cases of viral encephalitis (22%), 2 cases of Dengue (2%), 2 cases of Cerebral malaria (2%) and 5 cases of viral ADEM (5%).

It was observed that CNS infections were the commonest cause of non-traumatic coma. This is also supported by other studies, wherein infections of the CNS were found to be the leading causes of non-traumatic coma in children [12, 13].

Of the neuro infections, Pyogenic meningitis constituted the most common accounting for about 52.5%. This is similar to a study done by Awasthi S et el [8], Department of Paediatrics, King George's Medical College, Lucknow, Uttar Pradesh, where 42.2% had pyogenic meningitis out of 230 cases with acute CNS

infection.

Among the non-infectious causes, toxic-metabolic causes (no- 35, 18%) were the commonest and were also comparable in frequency with other studies [3,5,12,13].

Encephalopathy accounts for about 59 cases (32.7%) which included Toxic (16 cases – 8%), Metabolic (19 cases – 10%), Hypoxic (21 cases – 11.6%). 3 cases were due to hypertensive encephalopathy (1.6%).

Etiology of toxic encephalopathy included neem oil (no-8), Drugs (no-5) including carbamazepine(no-3), antipsychotic drugs like reserpine(no-1), INH(no-1), organophosphorus compounds (no- 2), Cypermethrin (no- 1).

Toxic encephalopathy included a case of accidental INH poisoning which presented with seizures and coma, the child was treated symptomatically with anticonvulsants and Pyridoxine and the child regained consciouness within 12 hours [16].

Neem oil encephalopathy is one of the important cause for toxic encephalopathy in Tamilnadu. In a study conducted in our

institute by Dr. Suresh during the period of 2005 to 2007, there were 88 cases of neem oil encephalopathy of which 27 cases died.

In a study conducted by Nayana P C Praba et el [10] on Role of Glascow coma scale in pediatric nontraumatic coma in JIPMER reported that 8 out of 218 cases of coma were due to toxic encephalopathy including neem oil.

Metabolic causes included hepatic and diabetic coma. Hepatic encephalopathy constituted about 15 cases of which 3 were due to Wilson disease and 2 were due to Leptospirosis.

Hypoxic causes of encephalopathy were due to status epilepticus (no-18, 10%), neuro-paralytic snake bite, cobra (no-1, 0.5%), near drowning (no- 2, 1%).

Hypertensive encephalopathy constituted about 3 cases of which 2 is due to chronic renal failure, 1 is due to acute glomerulonephritis.

17 (9.4%) cases are of vascular etiology which included

Intracranial Hemorrhage, thrombosis and infarction.

The etiology of coma was not known in 2.7% (no- 5) of the cases.

Similar study done by Arun bansal at PGIMER [7], Chandigarh showed the etiology of coma in 60% cases was CNS infection (tuberculous meningitis-19, encephalitis-18, bacterial meningitis-16, others-7); other causes were toxic-metabolic conditions (19%), status epilepticus (10%), intracranial bleed (7%), and miscellaneous (4%).

SOFIAH A et el and HUSSAIN I. H. M. I et el [3] at Neurology Unit, Paediatric Institute, Kuala Lumpur Hospital, MALAYSIA showed that the etiology of coma in the 116 cases they followed, eighty cases (69%) were due to infection, 15 (13%) to toxic metabolic causes, six (5%) to hypoxic ischaemic insults, four (3.5%) had intracranial haemorrhage, nine (7.8%) were due to miscellaneous causes and in two (1.7%) the cause was unknown.

CLINICAL PROFILE AND OUTCOME

OUTCOME:

Among the 180 cases studied, 78 (43.3%) survived and 102 (56.7%) cases died. Of the 102 cases, 36 died within 24 hrs of admission, 55 died between 24- 48 hours and 11cases died after 48 hours.

Of the 72 cases survived, 63 were discharged without any sequelae and 15 cases had sequelae. The sequelae included hemiparesis (3), PVS (5), rigidity & extrapyramidal movements (2), hydrocephalus (3), aphasia (1), cortical blindness (1).

AGE AND GENDER:

51 were less than 1 yr of age, 86 were between 1 to 5 yrs, 43 were between 6 to 12 yrs with a mean age of 3.46 yrs. Of the 180 children, 106 (58.9%) were males and 74 (41.1%) were females.

The mortality rate was higher in children <5 years age group and there is no statistically significant relation between the gender

and outcome. The higher incidence and mortality in children <5 years age group is related to higher frequency of toxic-metabolic causes, intracranial bleed and higher mortality with CNS infections. The incidence and outcome of coma was not associated with gender.

Seshia and Seshiaet el also did not observe any significant difference in the incidence of coma between the two sexes. Earlier studies [21] had shown a greater mortality in male (42%) compared to female children (20%)

ETIOLOGY AND OUTCOME:

The mortality was highest among Intracranial infection (57.8%, 59/102). The mortality rate among vascular and encephalopathies were 9.8% (10/102) and 30.5% (31/102) respectively.

The case fatality rate was 59.5% (59/99) for intracranial infection, 58.8% (10/17) for vascular etiology , 52.5%(31/59) for encephalopathies, and 40% (2/5) for idiopathic coma.

Among the intracranial infection, mortality rate was higher in Pyogenic meningitis (39/52, 75%). The mortality was also equally higher in Cerebral malaria(1/2) and Dengue encephalopathy (1/2).

In the viral encephalitis group, there were 2 cases of herpes encephalitis (characteristic findings in CT & MRI), but only one case survived among the two. The one who survived had sequelae (Aphasia, drooling of saliva). But on follow-up the patient improved, drooling of saliva stopped by 3 months and the patient regained speech by 1 year [25].

Among the toxic encephalopathies, neem oil constituted a major group (8/16 cases) and the mortality was 50% (4/8).

The mortality was also high in hepatic encephalopathy (10/15, 66.6%). It also included three cases of Wilson disease.

The mortality in hypoxic encephalopathy was 62% (13/21). It included a case of near drowning [27].

The mortality rate among the intracranial bleed was 69% (9/13).

In our study we have noted that etiology did not affect the

outcome. Similar observation was made by Nayana P C Praba in their study[10]. But many authors [17,22,23] have concluded in their study that outcome of coma was dependent on etiology.

It is important to realize that death often results not only from the primary neurological problems, but also from other secondary non-neurological causes. Therefore these could have affected the assessment of the effect of etiology on outcome in our study.

MGCS:

The MGCS at 24 hours was < 8 in 160 patients and between 9- 12 in 15 and between 13 – 15 in 5 patients with a mean score of 5.37. The MGCS at 48 hours was <8 in76, between 9- 12 in 30 and >12 in 19 with a mean score of 7.61. Of the 160 with score < 8 at 24 hours, only 62 survived with a mortality rate of 61% (98/160). The mortality rate for those with score <8 at 48 hours was 61% (46/76). Death was more common in cases with lower MGCS scores at 24 hr and 48 hrs. The prognosis was good for those with higher scores at the time of admission.

This is similar to a study conducted by Pushpa Chadurvedi et el and Manu kishore et el [15] at Mahatma Gandhi institute of medical sciences, Wardha on Modified Glasgow Coma Scale to predict mortality in febrile unconscious child, where the positive predictive value for death for those with lower scores (MGCS <5) was 88.8%.Many earlier studies also reported similar findings[2,5,6,7,8,9].

BRAINSTEM FUNCTIONS

1) PUPILLARY REFLEX:

Of the 180 studied, pupillary reflexes were preserved in 136 patients and absent in 44 cases. The mortality rate for those with non-reactive pupils was 88.6% (39/44). However, it should be appreciated that about 11% of children with non-reactive pupils have survived in our study.

Pupillary signs were very good predictors of survival and neurologic outcome. Non-reactive pupils at admission as well as at

48 hours were strong predictors of a fatal outcome. Similar observation was made in a study by Arun Bansal et el [7].

In the study by Seshia et al 68% of children with fixed dilated pupils for more than 2 hr died [5].

Ogunmekan made similar observations in a large retrospective study from Nigeria [13].

2) BRAINSTEM REFLEXES:

Presence of Doll's eye movements suggests intact interconnections between cranial nerve nuclei III, IV and VI via the medial longitudinal fasciculus and intact vestibular input to this system. Asymmetrical or partial absence of eye movements therefore, generally indicates asymmetrical brainstem lesion in mid brain or pons while complete absence of doll's eye movements suggests bilateral structural brainstem abnormality or severe metabolic-toxic encephalopathies.

In our study brainstem reflexes were preserved in 61.1% of the cases

(110/180). The mortality rate for those with absent brainstem reflexes was 84.3% with a relative risk of 3.88.

Nayana P C Praba et el in their study also noted relationship between brain stem reflexes and poor outcome.

Various other authors also have noted similar findings [4, 5, 7,10, 13, 14].

DURATION OF COMA AND OUTCOME:

In our study the mean duration of coma did not affect the outcome in terms of mortality. But there is a high incidence of sequelae in these patients. Similar observation was found in the studies by Pushpa Chadurvedi et el [15] and Aswati S et el [8]. But this is in contrast to a study by Quzi S A, Shan M A, Mughal N et el [24]

In our study we also found 89% (91/102) of the deaths occurred within first 48 hours of admission. These cases could be identified on admission with the help of MGCS. Similar observation was found in a study by Pushpa

Chadurvedi etel [15] where 85.7% of the deaths have occurred in first 24 hours of admission.

CONCLUSIONS

- The incidence of coma is high in age less than 5 years.
- The incidence of coma is high in males when compared to females.
- Intracranial infection is the most common cause of coma in children.
- Mortality rate in this study was 56.7% (102/180).
- 20.8% had sequelae.
- Age have an association with mortality.
- The mortality was highest in the intracranial infection.
- Gender did not have an association with outcome.
- MGCS scores can be easily assessed at bedside without any investigations. It shows a definite association with

mortality.

- Low total MGCS score was associated with adverse outcome. Mortality was high in those with MGCS score <8, when compared to those with a score >8.

- The prognosis was better for those with higher scores at the time of admission.
- Absence of brain stem reflexes predicted adverse outcome.
- There is no association between duration of coma and mortality.

LIMITATIONS OF THIS STUDY

- Etiology could not be found out for those with shorter duration of hospital stay.
- Follow up could not be done to predict long term outcome.
- Certain metabolic conditions (IEM, Reye syndrome) could not be ruled out because of limited investigation facilities.
- EEG could not be done in all the cases.
- Head injury cases are not included in our study as they were not managed in our department.
- Toxicological studies in cases with toxic encephalopathy could not be done due to practical difficulties.
- Radio imaging (CT, MRI) could not be done in all cases due to poor general condition or financial constraints
- Ventilator therapy could not be guided by ABG analysis due to lack of facility.

- Microbiological confirmation of acute CNS infection were not possible for majority of the cases due to technical difficulties in our setup.

RECOMMENDATIONS

- All the investigations for complete work up should be available in tertiary care hospitals.
- Nursing staffs and students can be taught MGCS, so that monitoring the progress can be easier.
- Early recognition and intensive management of shock and respiratory depression may improve the survival of the child.
- The supportive facilities for management of coma such as vital sign monitors, appropriate biochemical investigations and electrophysiological studies (bedside EEG in cases of refractory status epilepticus) needs further improvement.

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ABBREVIATIONS

CNS	-	Central Nervous system
MGCS	-	Modified Glasgow Coma Scale
ICT	-	Intra cranial tension
PPRF	-	Para Pontine Reticular Formation
PVS	-	Persistent vegetative state
ADEM	-	Acute disseminated encephalomyelitis
INH	-	Isoniazid
CT	-	Computed Tomography
MRI	-	Magnetic Resonance Imaging
MRA	-	Magnetic Resonance Angiography
TFT	--	Thyroid Function Test
ESR	-	Erythrocyte sedimentation rate
CRP	-	C reactive protein
ABG	-	Arterial blood gas analysis
ICP	-	Intracranial pressure
PCR	-	Polymerase chain reaction

MASTER CHART ABBREVIATIONS

SEX 1 - MALE
2 - FEMALE

BRAINSTEM REFLEXES AND PUPILLARY REFLEX
1 - PRESERVED
2 - ABSENT

ICT
1 - NO ICT
2 - PRESENT

DIAGNOSIS

1 - INTRACRANIAL INFECTION
2 - ASCULAR
3 - ENCEPHALOPATHIES
4 - IDIOPATHIC

ETIOLOGY, CLINICAL PROFILE AND OUTCOME OF COMA IN CHILDREN

NAME : AGE/SEX :

ADDRESS : OCCUPATION/INCOME:

PRESENTING COMPLAINTS :

HISTORY OF PRESENTING COMPLAINTS:

C/O UNCONSCIOUSNESS

C/O CONVULSIONS

C/O FEVER

C/O VOMITING

H/O ALTERED SENSORIUM

H/O ALTERED SLEEP PATTERN/BEHAVIOUR

H/O BLEEDING TENDENCIES

H/O DRUG INTAKE

H/O JAUNDICE

H/O ABNORMAL ODOUR IN BREATH

H/O ABNORMAL ODOUR/DISCOLOURATION OF URINE

H/O OLIGURIA/ANURIA

H/O DIARRHOEA

H/O EAR DISCHARGE

H/O RASHES

H/O LOSS OF WEIGHT/ APPETITE

H/O TOXIN INGESTION

H/O PALPITATIONS/CHEST PAIN

H/O COUGH

H/O HEADACHE

H/O DROWNING
H/O SWEATING

PAST HISTORY

ANTENATAL HISTORY

NATAL & POSTNATAL HISTORY

DEVELOPMENTAL HISTORY

IMMUNISATION HISTORY

FAMILY HISTORY

CONTACT HISTORY

GENERAL EXAMINATION

VITALS
HR
RR
BP
CRT
TEMPERATURE

ANTHROPOMETRY
HEIGHT/LENGTH
WEIGHT
HC
CC
AG
MAC

CHILD FEBRILE
HYDRATION
PALLOR
ICTERUS
CYANOSIS
CLUBBING
GENERALISED LYMPHADENOPATHY
ABD DISTENSION
PEDAL EDEMA
ABNORMAL ODOUR
NEUROCUTANEOUS MARKERS
ANTERIOR FONTANELLAE
SIGNS OF LIVER CELL FAILURE

FOCI OF SEPSIS – impetigenous skin lesions
bone/joint involvement
EXAMINATION OF EAR – discharge/ perforation

EXAMINATION OF CNS

LEVEL OF CONSCIOUSNESS

MGCS:

TIME	0 hrs	12 hrs	24 hrs	48 hrs
SCORE				

EXAMINATION OF CRANIAL NERVES

MOTOR SYSTEM RIGHT LEFT

BULK
TONE UL
LL

POWER UL
LL
DTR
JAWJERK
RIGHT LEFT
UL BICEPS
TRICEPS
SUPINATOR

LL KNEE
ANKLE
SUPERFICIAL REFLEXES
CORNEAL
CONJUNCTIVAL
ABDOMINAL
PLANTAR

SENSORY SYSTEM
CEREBELLAR SIGNS
MENINGEAL SIGNS
INVOLUNTARY MOVEMENTS
BRAINSTEM REFLEXES DOLLS EYE REFLEX
FUNDUS

EXAMINATION OF CVS

APICAL IMPULSE
HEART SOUNDS
MURMURS

EXAMINATION OF RS

SHAPE OF THE CHEST
AIR ENTRY
BREATH SOUNDS
ADDED SOUNDS

EXAMINATION OF ABDOMEN

SHAPE OF THE ABDOMEN
LIVER
SPLEEN
BS
FREE FLUIDS
EXTERNAL GENETALIA

INVESTIGATIONS

BLOOD

TC

- DC
- ESR
- HB
- PERIPHERAL SMEAR
- COMPLETE HEMOGRAM
- PLATELET COUNT
- BLEEDING TIME
- CLOTTING TIME

URINE
- ALBUMIN
- SUGAR
- DEPOSITS
- BILE SALTS, PIGMENTS

BLOOD UREA
- SUGAR
- CREATININE

LFT
- Sr. BILIRUBIN TOTAL
  CONJ
  UNCONJ
- Sr. ALK PHOSPHATASE
- SGOT
- SGPT
- Sr. AMYLASE
- Sr.PROTEINS TOTAL
  ALBUMIN
  GLOBULIN

SERUM ELECTROLYTES

CEREBROSPINAL FLUID ANALYSIS
- CELL COUNT
- BIOCHEM ANALYSIS
  - SUGAR
  - PROTEINS
  - CHLORIDES
  - GLOBULINS
- GRAM STAINING
- AFB STAINING
- CULTURE & SENSITIVITY

MANTOUX

SPUTUM/GASTRIC JUICE FOR AFB

CHEST X RAY

ECG

ECHO

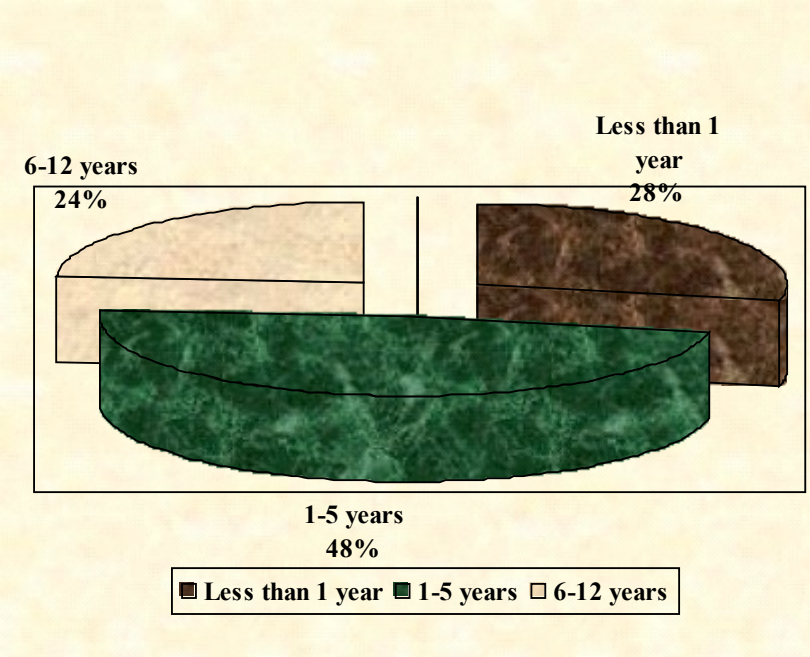
USG ABDOMEN

CT BRAIN

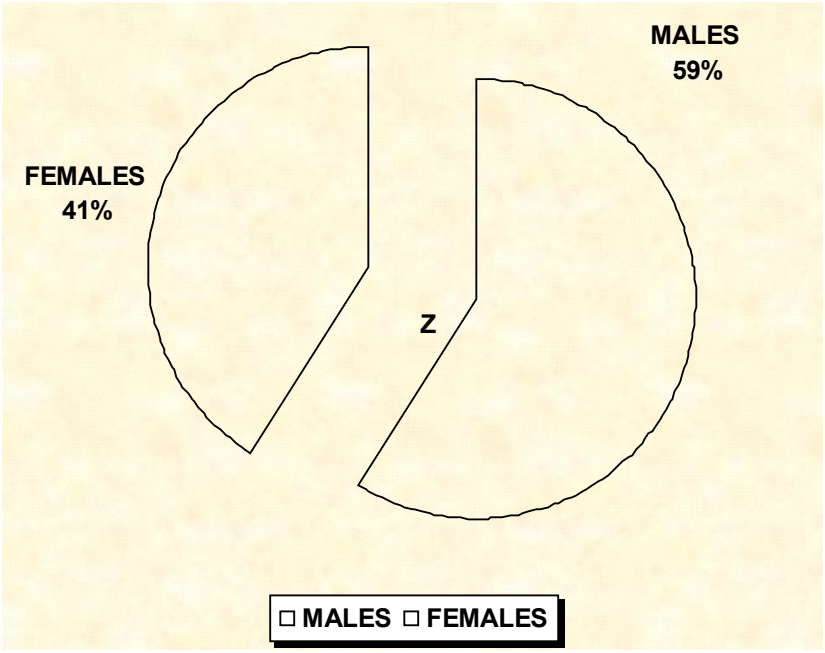
MRI BRAIN

URINE METABOLIC SCREENING

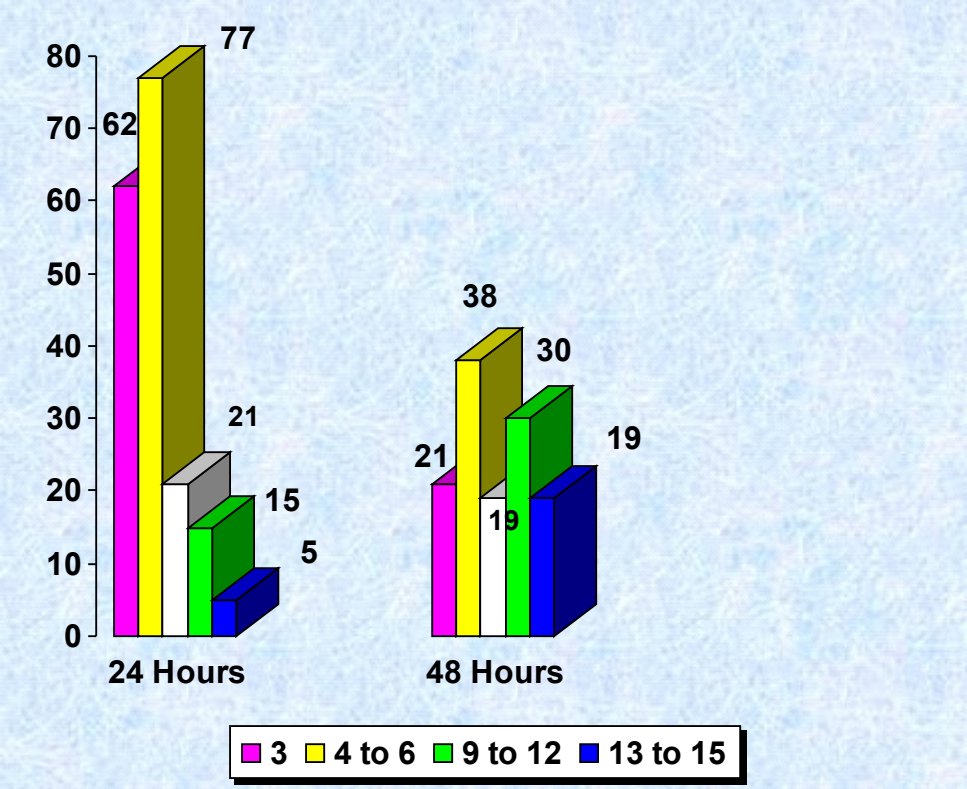
AGE DISTRIBUTION



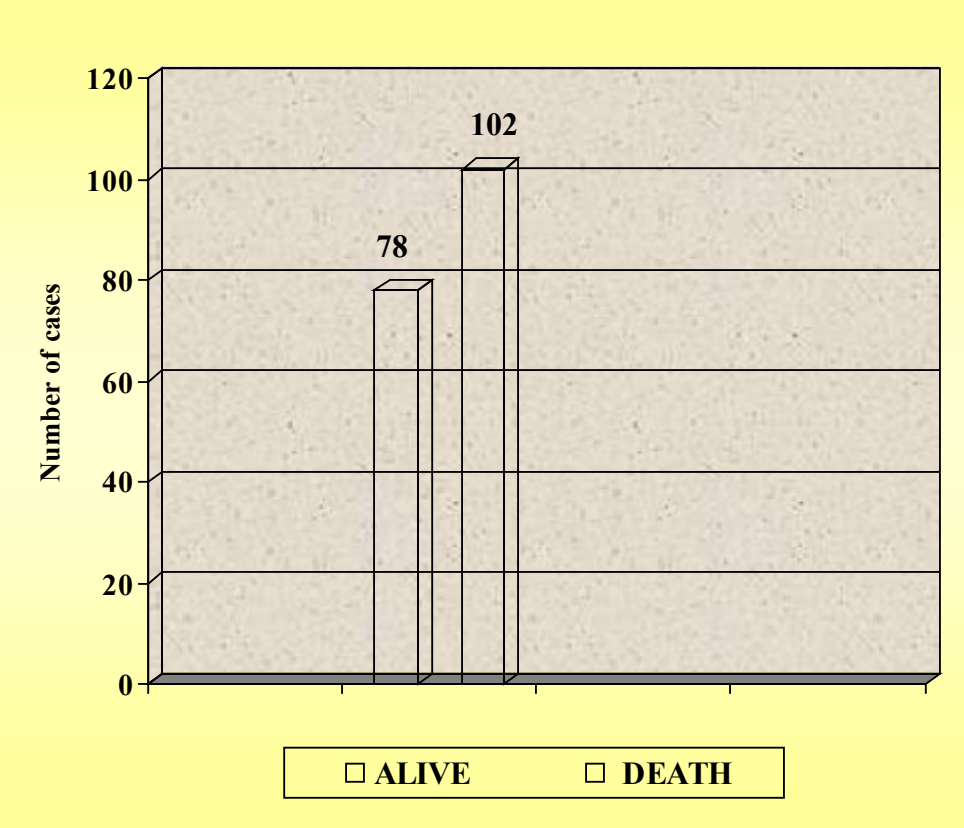
GENDER DISTRIBUTION



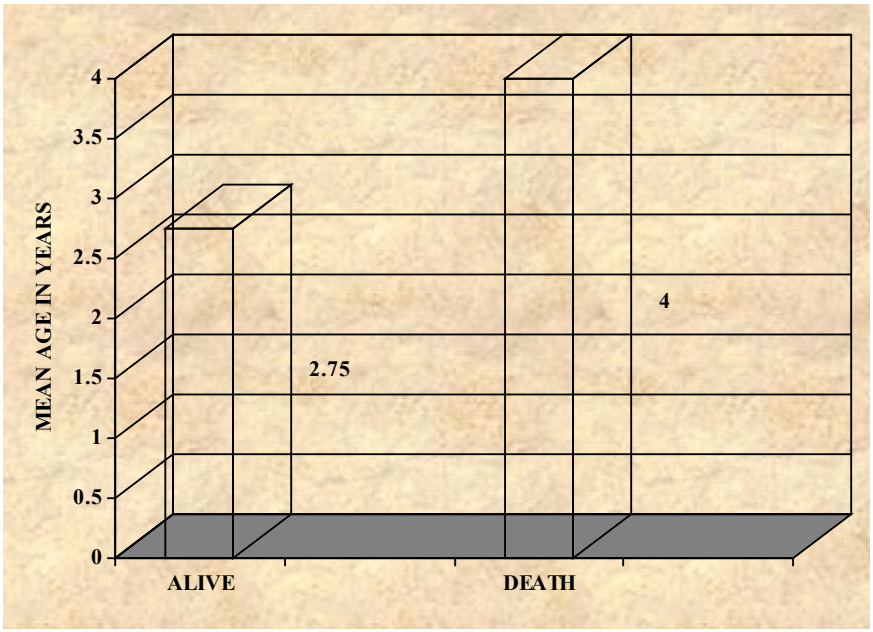
MGCS AT 24 & 48 HOURS



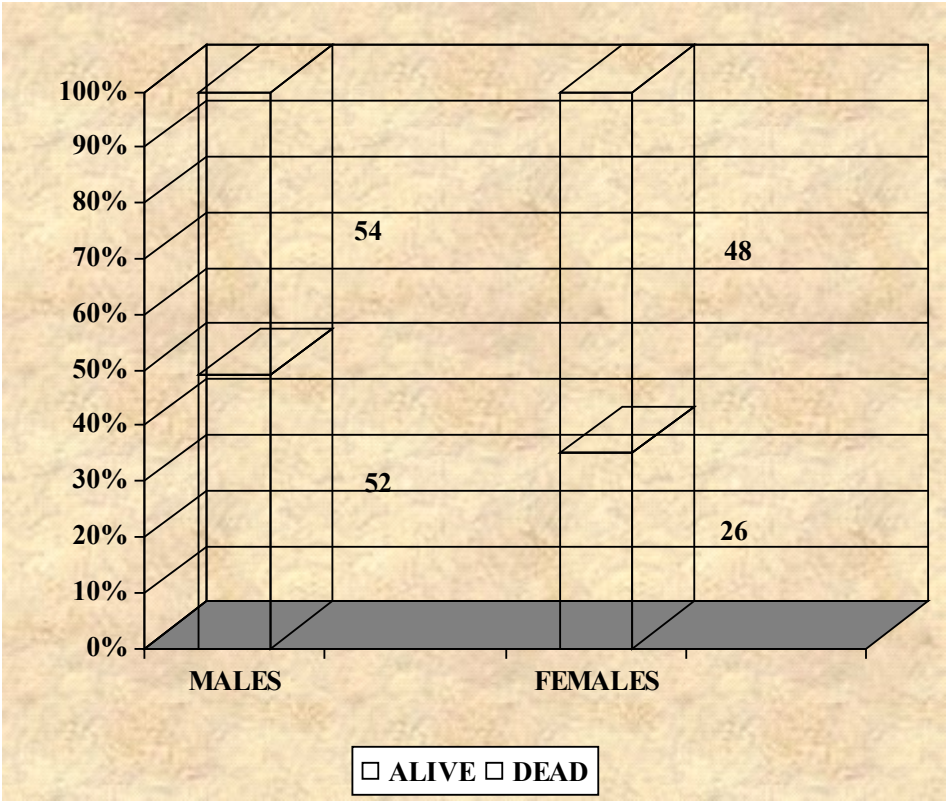
OUTCOME



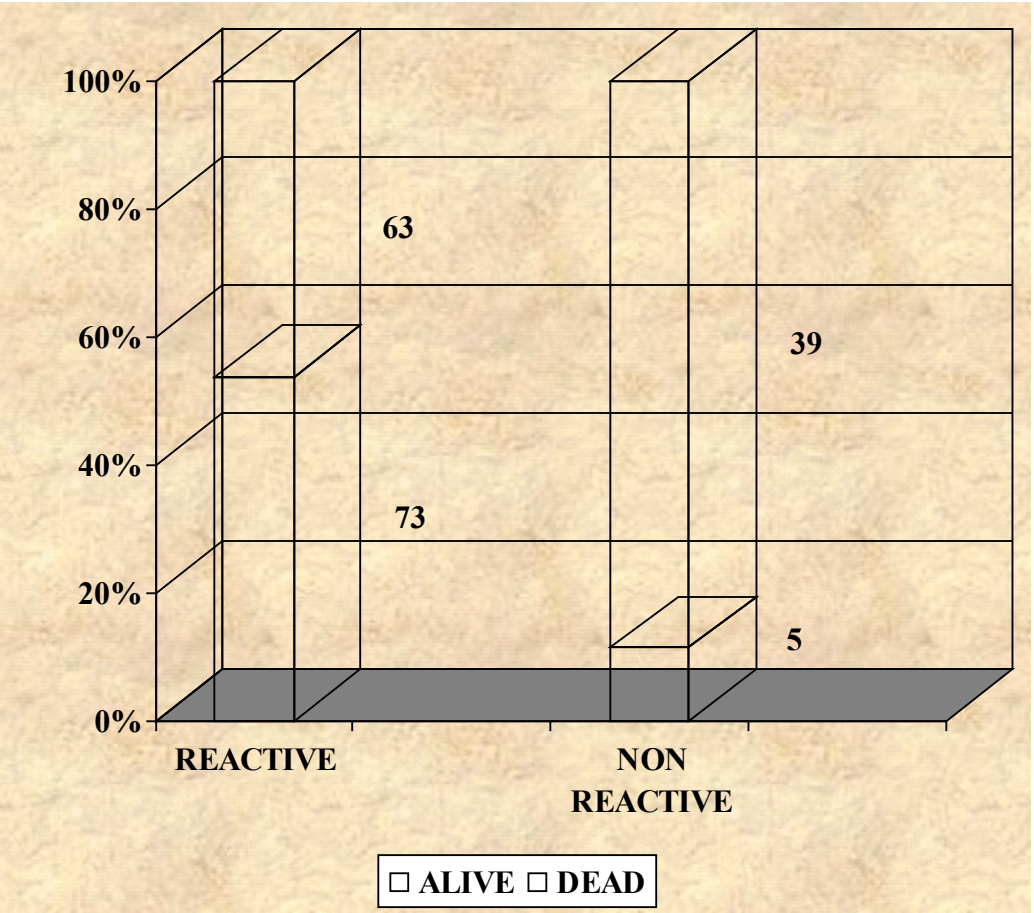
AGE AND OUTCOME



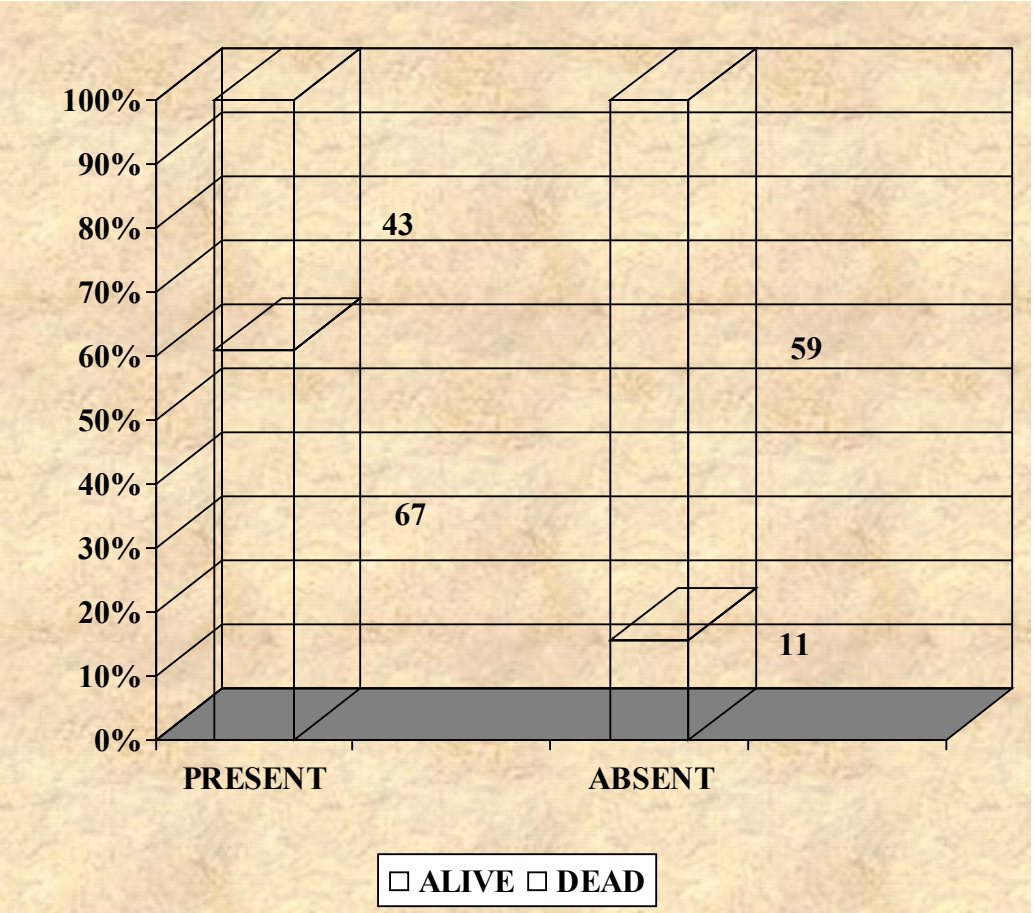
GENDER AND OUTCOME



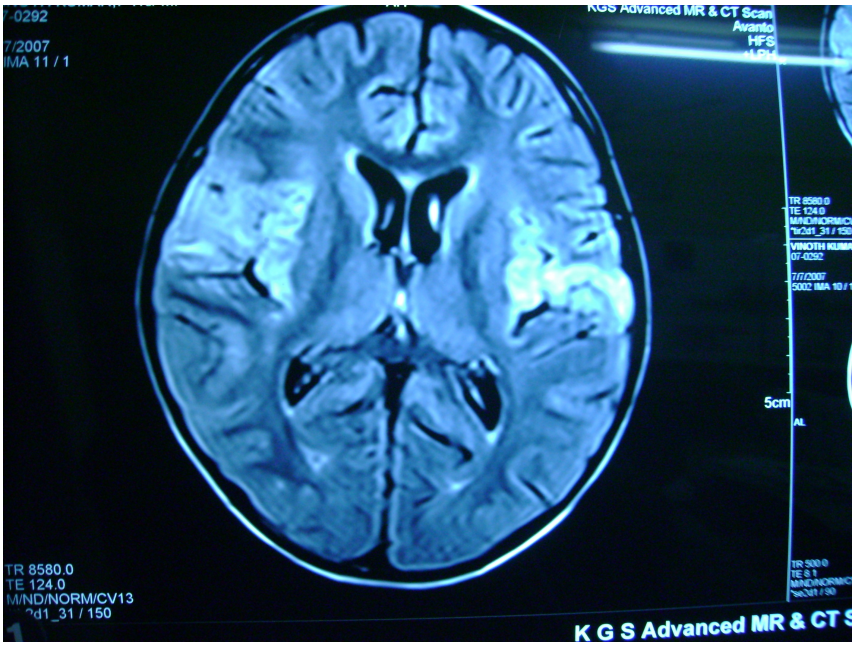
PUPILLARY REFLEX AND OUTCOME:



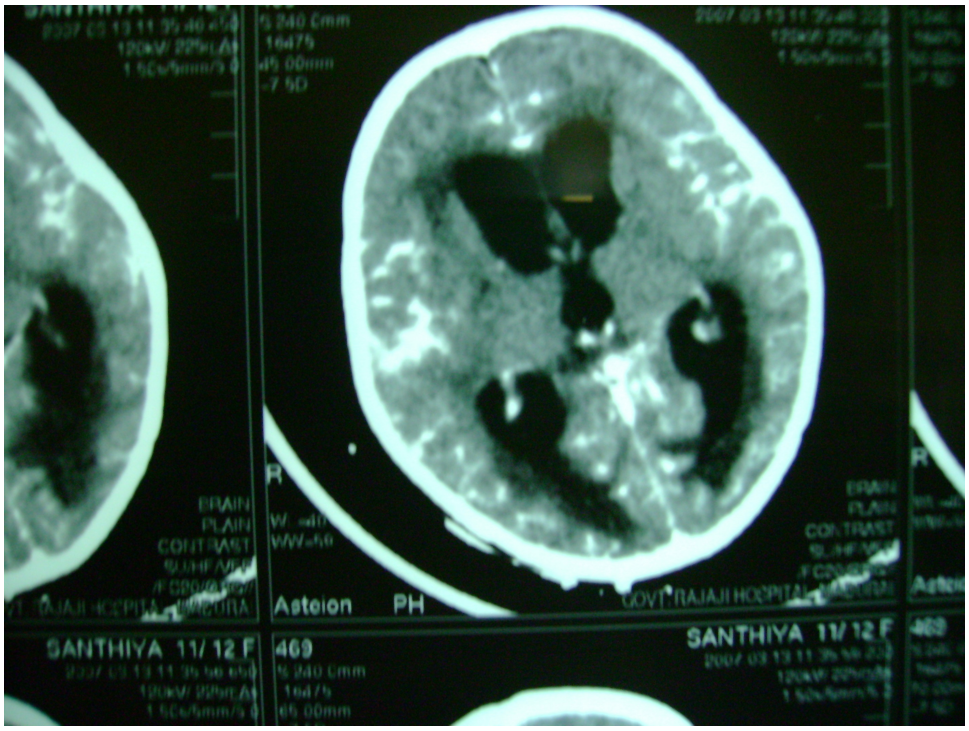
BRAINSTEM REFLEXES AND OUTCOME:



HERPES ENCEPHALITIS



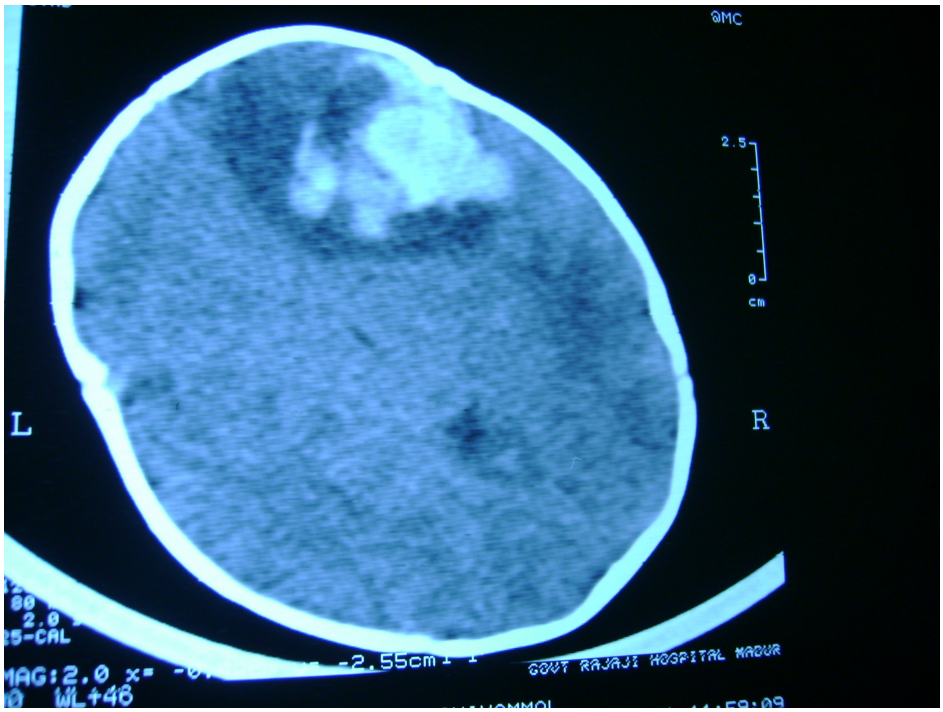
TUBERCULOUS MENINGOENCEPHALITIS WITH OBSTRUCTIVE HYDROCEPHALUS



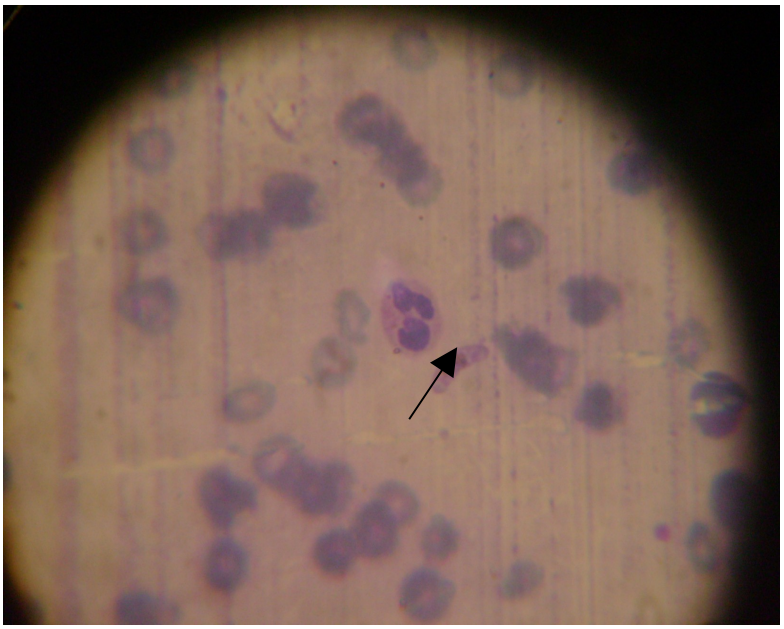
COMATOSE CHILD

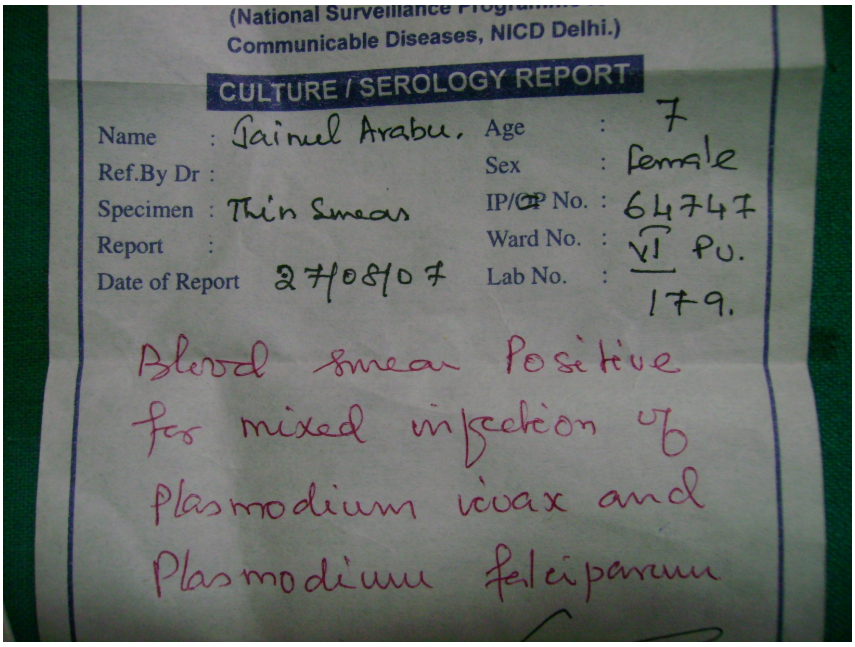
CORTICAL VENOUS THROMBOSIS WITH "EMPTY DELTA SIGN"

HEMORRHAGE



CEREBRAL MALARIA





(National Surveillance Programme for
Communicable Diseases, NICD Delhi.)

CULTURE / SEROLOGY REPORT

Name : Jainul Arabu. Age : 7
Ref.By Dr : Sex : Female
Specimen : Thin Smear IP/OP No. : 64747
Report : Ward No. : VI Pu.
Date of Report 27/08/07 Lab No. : 179.

Blood smear Positive for mixed infection of Plasmodium vivax and Plasmodium falciparum